PAPERS AND ORIGINALS

Long-term outcome after severe head injury

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Summary

From a consecutive series of 7000 patients with head injuries admitted to the regional accident service, Radcliffe Infirmary, Oxford between 10 and 24 years earlier, every patient was taken who had been amnesic or unconscious for one week or longer. Of these 479 patients, all but ten were traced, and either the cause of death was established or the survivors examined. Ten years after injury 4% were totally disabled, and 14% severely disabled to a degree precluding normal occupational or social life. Of the remainder, 49% had recovered, and the rest were dead. Additionally, a selected series of 64 patients whose unconsciousness had been prolonged for a month or more were studied. Forty of these had survived between three and 25 years after injury and were re-examined.

On the basis of age at injury, the worst state of neurological responsiveness, and the duration of post-traumatic amnesia, the outcome of head injury can be predicted reliably in most cases. Patients and relatives need more reassurance and simple psychotherapeutic support, especially in the first few months after injury. Extrapolation from our figures suggests that each year in England and Wales 210 patients survive totally disabled and another 1500 are severely disabled.

Introduction

Most of those who survive the severest forms of accidental head injury are healthy adolescents and young adults with a further life expectancy of half a century or more. Improvements in resuscitation and treatment of some of the complications have salvaged progressively larger numbers of these patients. The extent to which various disabilities due to brain damage after closed head injury resolve or are modified in the long term is not clear, neither is how often and in what form these disabilities persist. Existing data tend to be conflicting since they are based on heterogeneous series of patients. In viewing the effect of brain damage in head injury, it is easier to see in retrospect the evolution of a pattern of disability if we can compare information about the acute and convalescent stages with the state of the patient examined years later. In this way the distracting problems of acute management are avoided.

Prognosis is related to age at the time of injury,¹⁻³ the severity of the injury as judged by indirect measures of the worst level of neurological responsiveness after injury,⁴⁻⁷ the duration of decerebration,^{8,9} altered consciousness,¹⁰⁻¹⁶ post-traumatic amnesia,¹⁷ and the rate of recovery from neurophysical disability in convalescence.¹⁸⁻²³ These earlier studies, however, used no consistent definitions either of the severe injury or of the "long term."

Patients and methods

Every patient amnesic or unconscious for one week or longer was identified in a population of 7000 admitted consecutively, between 10 and 24 years earlier, to the regional accident service, Radcliffe Infirmary, Oxford, which provided neurosurgical care of patients with head injuries. All but 10 of these 479 were traced, the cause of death of 178 was established, and the remaining 291 were followed up and re-examined. To enlarge the most severely injured group a further selected series of 64 patients, taken from other sources, who had survived injuries which caused unconsciousness lasting for a month or more between three and 25 years earlier were also studied. The cause of death of 28 of these was established, one was untraceable, and the remaining 40 were re-examined. Details of acute and convalescent stages after injury were abstracted from the hospital case records. Each patient underwent a neurological examination by one of us (AHR), and 217 (about two-thirds) were then given a selected series of tests of cognitive function. Independent accounts

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were provided by one or more relatives for 90% of those examined. Age at injury and follow-up are shown in table I. The severity of the injury using various indices and the proportions of patients so injured are shown in fig 1. The management of these patients has been described by one of us (WL).²⁴

Injury had been complicated by brain compression or brain penetration, either traumatic or surgical for internal decompression, in 77 cases of the consecutive series and in 14 cases of the selected series who survived to be followed up.

Unconsciousness was defined as persisting until there was comprehension of the spoken word as demonstrated by obeying a verbal request.

Decerebration was shown by a response to stimulation consisting of extension of one or both legs with extension or flexion of one or both arms, and was defined as persisting until purposive or semi-purposive movements were recorded.

Post-traumatic amnesia (PTA) was assumed to last until there was restoration of orientation and recall of day-to-day events.

TABLE I—Ages at injury and follow-up of 331 patients with severe head injuries. Figures are numbers (%) of patients

Age (years):		5-15	16-25	26-35	36-45	46-55	56+
<i>Consecutive series (n=291)</i>							
At injury		48 (17)	101 (35)	52 (18)	40 (14)	36 (12)	14 (5)
At follow-up		31 (11)	75 (26)	76 (26)	37 (13)	72 (25)	
<i>Selected series (n=64)*</i>							
At injury		13 (20)	38 (60)	4 (6)	5 (8)	4 (6)	
At follow-up		4 (6)	14 (22)	25 (39)	12 (19)	6 (9)	3 (5)

*Includes 24 patients from consecutive series.

FIG 1—Severity of head injury in survivors followed up. Top: consecutive series of 291 patients in whom post-traumatic amnesia (PTA) lasted one week or longer. Bottom: selected series of 64 patients, in whom prolonged unconsciousness lasted one month or longer. Figures above columns refer to numbers of patients. Selected series includes 24 patients from consecutive series.

Disability profiles were scored on a ten-point scale for various components of the central neural disability (CND): akinesia, imbalance, ataxia, paresis, and sensory deficit; and for mental disability (MD) for frontal-lobe personality change, irritability, anxiety including depression, obsessionalism, paranoid ideation or behaviour, memory impairment, and dysphasia. The same scale was used to assess the initial level of neurological responsiveness, progress, and final disability.

The grades of neural dysfunction in the acute and convalescent stages, and disability later, were scored separately for neurophysical and mental effects: 5, decerebrate response/mindless dementia; 4.5, purposive/semipurposive response; 4, bed or chair bound/delirious, inaccessibly demented or psychotic; 3.5, ambulant with assistance/accessibly demented or psychotic; 3, profound akinesia, hemiparesis, ataxia but walking unassisted/personality change, dementia, dysphasia or psychiatrically ill to an extent precluding normal domestic, social, or occupational life; 2.5, the same, severely limiting but not precluding normal activity in these spheres; 2, the same, causing moderate difficulties at home, work, or socially; 1.5, the same, causing only inconvenience; 1, the same, of which the patient or relative is aware, and detectable clinically but of minimal importance; 0.5, detectable only on examination and causing no disability; and 0, none.

Results

We recognised four different clinical syndromes of neurological lesions, and eight less clearly defined patterns of mental disability. These persist indefinitely after a single, severe, closed head injury. They are discussed in detail elsewhere.²⁵ The descriptive titles here draw attention to features of the mental and neural disorder and to the sites of brain injury these imply.²⁶⁻³⁰

NEUROPHYSICAL DISABILITY

In *decerebrate dementia* decerebrate reflexes were the principal response to stimulation, though semi-purposive movement developed on one side of the body in a few cases many months after injury. There was usually no detectable intellectual function. In the consecutive series no patient in this category was still alive more than ten years after injury.

In the *athetoid pseudobulbar* pattern of lesion (5% of the consecutive series) there was severe bilateral pyramidal damage with postural dystonia and often bradykinesia and fragmentary athetosis. A few patients showed remarkable discrepancy between the severity of the physical disability and the relative preservation of personality and intellectual function, suggesting that focal injury by secondary ischaemic effects caused this pattern as well as primary traumatic damage.

In the *brain-stem cerebellar* pattern (20% of the consecutive series) the most striking feature was the evidence of asymmetrical cerebellar and pyramidal lesions. Damage was clearly not confined to the brain stem, especially since frontal personality changes were often apparent, but this condition reflects the lesions found extensively throughout the brain stem either directly because of trauma or indirectly because of compression from brain swelling.³¹

The *hemiparetic* was the commonest residual pattern (40% of the consecutive series) but signs of unilateral pyramidal injury were usually slight. Severe hemiparesis was usually associated with the preceding three patterns, and only then was cortical sensory loss commonly encountered. About 21% of the consecutive series had no detectable neural lesion, and 14% did not fall into any of these categories.

Life expectancy was closely related to the pattern of residual neural lesions. Most patients who survived in states of decerebrate dementia died within a year after injury, and though a few lived for several years, only one from the two series survived for a decade. In contrast, those with even the most severe forms of athetoid pseudobulbar syndrome who recovered sufficiently to be discharged from hospital and walk unassisted appeared to have little reduction of life expectancy except by epilepsy, accidental drowning, inhalation of food, and suicide. Those with varying degrees of the brain stem cerebellar or minor hemiparetic pattern of lesion who recovered from the immediate effect of their injury and left hospital had no more than an average reduction in life expectancy of five years. This seemed to be because death from causes uncommon in the general population occurred prematurely in a proportion of these survivors (table II),

TABLE II—Deaths from selected causes among 75 severely head-injured patients in the consecutive series who “recovered” sufficiently to be discharged compared with proportionate numbers dying from similar causes derived from death rates reported in 1960⁵⁰

Cause of death	Deaths among severely head injured	Deaths reduced pro rata on population of England and Wales	Increased risk of death after severe head injury	Significance
Meningitis	4	0.06	× 65	P < 0.001
Epilepsy	4	0.1	× 40	P < 0.001
Drowning	3	0.15	× 20	P < 0.001
Accidents	7	2	× 3	NS
Suicide	3	1	× 3	NS
Respiratory disease	17	8	× 2	P < 0.005
Cardiovascular disease	16	16	Same	
Malignant disease	11	14	Slightly less	
Cerebrovascular	8	10	Slightly less	

NS = Not significant.

and not because, as suggested,³² the brain damage itself exerted some specific effect on life expectancy by increasing the vulnerability of the body to the stresses of aging.

The degree of disability associated with each of these syndromes of central neural lesions was relatively constant (table III). Patients with decerebrate dementia or the athetoid pseudobulbar syndrome required continuing care in hospital, or in homes, or from relatives. Severe ataxia, euphoria, and disinhibition did not preclude a tolerable domestic or social life in many with the brain-stem cerebellar pattern. Those left with slight pyramidal signs alone were not disabled physically, though some were by personality change and defective memory. Among these patients intellectual function tests were more impaired when the head impact had been right-sided.³³

MENTAL DISABILITY

As the study progressed, patterns of persisting mental disability also emerged, which (like the patterns of disabilities due to central neural lesions) were closely related to the severity of the injury and the age at which it had occurred.

The most commonly encountered severely disabling pattern, *fronto limbic dementia* (50 cases), was seen only after the severest forms of brain injury and was confined virtually to adolescents and young adults with the athetoid pseudobulbar and severe brain-stem cerebellar patterns of lesion. In these patients frontal personality changes were combined with severe impairment of day-to-day memory and the release of irritability and rage reactions.³⁴

Frontal dysmnesia (34 cases) and frontal personality change without released irritability or memory impairment (21 cases) were a common sequel to less severe injury in the young. Frontal personality change with pathological irritability (12 cases) and memory impairment and irritability without the personality changes of orbito-frontal injury (10 cases) were less common. Focal cognitive deficits, including dysphasia (six cases), were rare in the absence of dementia. Two highly characteristic sequels of injury after middle age were patterns of disability dominated by anxiety: *dysmnestic inadequacy* (22 cases) apparently reflecting a neurotic response to memory and other less obvious cognitive deficits, and *phobic imbalance*³⁵ (11 cases) a neurotic reaction to various forms of traumatic vestibular disorder. In neither of these last two conditions was it usual to find other than minimal signs of central neural lesions. These two long-term sequels of severe head injury were thus seen to bear a close resemblance to the

so called postconcussional syndrome shown after minor head injury. There were 35 patients with persistent *traumatic dysmnesia* alone—that is, without personality change or other than minimal evidence of neural lesions. This was a common long-term sequel of the relatively less severe injuries at all ages.

Schizophreniform psychosis³⁶ occurred in seven of the 291 patients of the consecutive series, but, because of its invariable association with dementia and inconstant relationship to the severity of injury, *traumatic paranoid dementia* may more appropriately describe the condition. Evidence that schizophreniform illness in the absence of dementia was likely to be precipitated by severe head injury was slight. In contrast, depressive illnesses with the characteristics of endogenous affective disorders were clearly precipitated by injury in 4% of the consecutive series of patients.

CRANIAL NERVE INJURY

The prevalence of permanent anosmia increased with age at injury, rising to almost half where injury had occurred over the age of 45. The relatively uncommon evidence of the persistence of other cranial nerve lesions is in keeping with previous reports.^{37–39} Visual-field defects due to optic-nerve and chiasmal lesions were found in 3% of cases; diplopia because of residual third-nerve palsies in 7% of cases, and because of lateral rectus weakness in 4% of cases, the prevalence of both conditions being slightly higher in complicated injuries. Neither visual-field defects nor diplopia constituted serious disability, and only four patients had needed surgical correction for diplopia. Persisting cranial nerve lesions were otherwise rare.

PITUITARY AND HYPOTHALAMIC DAMAGE

Impairment of pituitary or hypothalamic function was rare. In contrast, transient hypothalamic hyperphagia (16 cases) and diabetes insipidus (eight cases) noted in the hospital records of the consecutive series of patients was common, as others have found.^{40 41}

EPILEPSY

There was an overall prevalence of post-traumatic epilepsy of 28% in the consecutive series. This varied from 7% among those with an uncomplicated injury and a PTA of no more than a week to 61% among those whose injuries were complicated by brain compression and traumatic or surgical penetration of the brain. There was increasing prevalence of epilepsy with length of PTA in uncomplicated injury, as there was in both complicated and uncomplicated injuries with duration of coma. In the survivors re-examined from the consecutive series the epileptic attacks had become progressively less frequent in all but three patients. There were no features of their epilepsy in the early stages after injury that would have identified these three patients, whose post-traumatic epilepsy was not benign. The long-term course of post-traumatic epilepsy in accidental head injury thus appears to be very similar to that occurring after missile wounds of the brain.^{42 43} This contrasts with the differences between the neurophysical (CND) and mental (MD) disabilities after these two types of injury.

PREDICTING THE LONG-TERM OUTCOME

Using only the age at injury and two indirect indices of severity of injury (the worst state of neurological responsiveness after injury—that is, maximum CND and maximum MD score) and the duration

TABLE III—Residual neurophysical disability associated with each of the four patterns of central neural lesion found at follow-up. Figures are numbers (%) of patients

	Decerebrate dementia	Athetoid pseudobulbar	Brain-stem cerebellar	Hemiparetic	None	Not classifiable
Consecutive series (n = 291)						
No (%) in group	..	16 (5)	58 (20)	115 (40)	61 (21)	41 (14)
No (%) severely disabled or worse	..	13 (82)	14 (24)	6 (5)		2 (5)
No (%) moderately disabled or better	..	3 (18)	44 (76)	109 (95)	61 (100)	39 (95)
Selected series (n = 64)						
No (%) in group	4 (6)	32 (50)	22 (34)	6 (9)		
No (%) severely disabled or worse	4 (100)	28 (87)	9 (41)	5 (83)		
No (%) moderately disabled or better	..	4 (13)	13 (59)	1 (17)		

of PTA gave a generally accurate guide to long-term outcome. These indices, which are usually available in even the least well-documented hospital records or in the case of PTA may be assessable in retrospect, were used sequentially in a computer-assisted analysis to examine their relation to patient outcome. The procedure used in this method of prediction of outcome was drawn up for both CND and MD as flow diagrams (fig 2) for the consecutive series of patients. The

FIG 2—Predicting long-term outcome (for three classes A, B, and C*) for consecutive series of patients using sequentially age at injury, worst state of neural dysfunction (maximum central neural dysfunction (CND) or mental disorder (MD) score), and duration of post-traumatic amnesia (PTA). Top: CND. Bottom: MD.

*See text for classification of patient outcome A, B, and C and for grades of neural dysfunction.

confidence with which each outcome was predicted was designated "strong," "moderate," or "weak," based on an arbitrary assessment of the extent of correct classification using these data. The predictions A, B, and C correspond to the three classes of final disability: A score 0.5 or 1 (minimal, negligible, or no final disability), B score 1.5 or 2 (mild or moderate), and C score 2.5 or more (severe or worse). The paths through the procedure were established by inspection of the data. All branches denote divisions into two groups with statistical significance of $P \leq 0.01$ (usually much less). The procedure allows for interaction between the three predictors.

Children whose injury has not caused decerebration will do well in the long term and be left with little or no neurophysical disabilities. If they have been decerebrate, most will still have little or only slight to moderate CND or MD unless the injury caused brain damage of a degree usually resulting in death. These cases will be only too easily identified within three months of injury. It follows that despite apparently profound degrees of impaired neural function in the acute stage after injury a poor prognosis of CND or MD should not be assumed within five years of injury for this group. Even then

the behavioural problems of adolescence may suggest a personality disorder that will not, however, persist.

Adolescents and young adults should all recover without CND if no more than confused after injury. There is a greater chance of slight to moderate memory impairment or personality change persisting, but this will rarely be severely disabling. Those patients whose level of neurological function after injury has been reduced to purposive or decerebrate response to stimulation and who have no more than two months' PTA and one day's decerebration will seldom be physically disabled. Those with longer PTA, decerebration, or both will be moderately or, more often, severely or profoundly disabled by their neural lesions. A PTA of two to eight weeks after similar degrees of disturbed neural function in the acute stage after injury will usually result in moderate disability by memory impairment or personality change.

The middle aged and older who are not decerebrate, and whose PTA lasts no more than a fortnight, will nearly all have no neuro-physical disability, but of those who are decerebrate most will have severe CND and MD. Decerebration for those aged 46 or older at injury is rarely survived very long. Persisting MD is commoner in this age group, but those patients only confused initially are unlikely to be severely disabled. Where the level of impairment of neural function after injury is reduced to purposive or semi-purposive response to stimulation, a PTA of up to one month will be seldom associated with more than moderate MD or CND. Such patients with more than one month's PTA will usually develop severe MD.

Some general statements can be made about final outcome in its relation to rate of recovery. No one walking unassisted within three and a half weeks of injury had any physical disability due to neural lesions in the long term. Decerebration lasting over two and a half weeks in children was compatible with little CND but in adults was invariably followed by severe and permanent MD or CND, or both, as also was coma lasting longer than a month when unassisted walking was delayed for three months after injury. Unassisted walking when delayed for three months was seldom succeeded by worse than moderate physical disability even in adults. Decerebration in adults and children for longer than three months invariably resulted in no recovery beyond vegetative or analogous states. Spasticity and moderate difficulty in using a limb persisted for up to 18 months in some cases but still improved thereafter so that there was finally negligible disability. Recovery from mental disorder lagged behind recovery from physical disorder and persisted and might cause moderate disability for up to two years in some cases yet thereafter resolved. Recovery allowing reclassification to a lesser grade of disability was still possible five years after injury for children but not for adults.

However accurate as an overall guide, these indices of long-term outcome did not take account of individual variability. The prerequisite of accurate long-term prediction for individual patients must be a first-hand experience of the mental and neurophysical disabilities which persist after severe acceleration brain injury. When patient progress is not as predicted an explanation should be sought in the individual's intelligence or personality before injury, focal ischaemic brain lesions superimposed on lesser overall mechanical damage, the separate problems of epilepsy and cranial nerve injury, unexpectedly precipitated psychiatric illness, and the question of simulation for gain.

OCCUPATION

Decline in occupational status was more strongly associated with MD than CND in all social classes with the exception of the unskilled manual worker. There was a progressively increasing risk of long-term unemployment with lengthening PTA and also with severer grades of impaired neural responsiveness after injury. Durations of PTA and the degree of impaired neurological function after injury were both closely related to time off work and failure to return to gainful employment. The likelihood that anyone will succeed in improving his occupational status after a head injury of this severity seems relatively small. Only 16 of the 291 patients of the consecutive series (6%) did so. In only one case was this after a PTA of over three weeks.

PROGRESSIVE DETERIORATION

There were no features of the head injury of those patients who became progressively demented that distinguished them from the

rest, yet progressive intellectual deterioration was seen in 31 cases (11%) of the consecutive series. This is twice the prevalence of dementia found in the community over the age of 65.⁴⁴ If there was a specific neuronal degeneration provoked by a single head injury it is unlikely that the pathology of this common condition would have been overlooked. Premature aging of damaged brain has been confirmed by comparing aging of elderly injured patients with those in a psychiatric community who had not sustained head injuries.⁴⁵

EPIDEMIOLOGICAL IMPLICATIONS

This study was designed to assess the long-term outcome of an equivalent of a random sample of severely head-injured patients from a clearly defined population admitted consecutively to one regional accident service providing neurosurgical care. It may be possible, therefore, to make some generalisations based on these results which apply to the population at large.

Field¹⁶ noted that a relatively constant 7000 to 8000 patients with head injuries remained yearly in hospital for two weeks or more before discharge or death. These represent 6% of an average 120 000 total number of patients admitted to hospital with head injuries in England and Wales annually between 1962 and 1972. Of 7000 patients admitted to the Radcliffe Infirmary, the 469 (6%) admitted with a PTA of a week or more selected for this study may be assumed to be similar to the 6% of 120 000 patients referred to above. One year after injury 13 (3%) of the 469 were totally disabled either mentally or physically and requiring constant care and attention, and 102 (22%) were severely disabled to a degree that either precluded or severely limited normal occupational, domestic, or social life. Two hundred and forty-seven (53%) had recovered, and the rest were dead. If these same percentages are applied to the 7000 patients remaining yearly in hospital for two weeks or more after head injury in England and Wales, at the end of the next year there will be 210 totally disabled survivors and 1500 severely disabled. These numbers will alter, because of death or further recovery, at ten years after the year of injury to 280 and 960 respectively. A similar cohort of disabled patients will survive head injury every year. Taking into account the near-normal life expectancy of the majority these figures suggest a substantial population requiring some degree of long-term care.

Tentative estimates can also be made of the numbers surviving yearly after head injury in England and Wales in states of dementia and decerebration. Applying our figures to the 7000 patients admitted to hospital yearly for two weeks or more after head injury in England and Wales suggests that there might be 42 surviving in these states by the end of the next year, 18 at three years, 12 at five years, and two at 10 years. Higashi *et al.*¹⁷ described similar proportions of survivors at these times in vegetative and analogous states after head injury but none for as long as 10 years. Survival for this length of time or longer is unlikely but does occur.⁴⁸⁻⁴⁹ It has been assumed that negligible numbers whose injuries resulted in PTA lasting under a week will remain severely disabled. This is justified because only six of 82 patients in our study whose PTA lasted under two weeks remained mentally disabled, severely physically disabled, or both in the long term.

These figures may be underestimates both of the numbers of severely disabled patients surviving in the past decade or so because of advances in resuscitation techniques and also of those with PTA lasting under a week who may have been more disabled than was suggested by this study of severer injuries. Even so the figures give some idea of the size and nature of the continuing problem of long-term disability after accidental head injury.

Conclusion

This study was concerned with the severest acceleration head injuries, yet severe physical or mental disability were uncommon. On the other hand, a few patients, because of their youth and modern techniques of resuscitation, survived such devastating brain damage that only the most insensitive would regard their survival as humane. For most patients, however, almost every effort will be worthwhile, especially in the young.

Even for those whose recovery was excellent the experience had been frightening and often disastrous. Most suffered weeks or months of misery, apprehensive of the significance of their forgetfulness, defective concentration, imbalance, and vertigo. Any personality change caused relatives intense distress.

What specific effects rehabilitation had was not evident, but the rehabilitation units available had clearly been helpful. There appeared to have been, especially with the less severely injured, an underestimate of the degree to which patients and relatives had been distressed by the effects of even briefly impaired intellectual function and personality change.

Greater efforts might be made to provide reassurance and simple psychotherapeutic support to patients and their relatives even for the least severely injured in the first few months. The disruptively aggressive patient must remain the responsibility of the psychiatric services because those less experienced in their management will give inadequate care. The more taxing problem of the few totally and permanently dependent patients cannot be solved except by establishing more units for the chronically sick. These are more appropriate than the geriatric wards or distraught families in which many were found.

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Has the mortality of male doctors improved with the reductions in their cigarette smoking?

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Summary and conclusions

From 1951 to 1971 male doctors reduced their cigarette smoking more than did men in social classes I and II combined. In 1970-2, 665 male doctors died aged under 65. Had they shown the same improvements in cause-specific death rates over the 20 years as men in classes I and II, 699 deaths would have been expected. This "saving" of 34 deaths in the doctors comprised savings from coronary heart disease (83), stroke (16), and lung cancer (8) balanced by 60 "losses" from three stress-related causes—namely, accidents, poisonings, etc (30); suicide (26); and cirrhosis of the liver (4)—plus 13 from other causes. As a relative reduction in mortality from heart disease in doctors (as compared with that in social classes I and II) also occurred during 1931-51—that is, before they began to give up smoking—some of the saving in heart-disease deaths in 1951-71 was probably not related to changes in smoking habits. The relative worsening in mortality from stress-related diseases may have been due partly to a possible adverse effect of giving up smoking if smoking had acted to reduce stress.

From these findings, the benefits of giving up smoking may not be so great as has commonly been assumed.

Introduction

Cigarette smokers who stop smoking are claimed to run a smaller risk of dying than if they continue to smoke.^{1 2} Much evidence quoted to support this comes from studies in which ex-smokers have been shown to have death rates intermediate between those of people who have never smoked and those of continuing smokers.^{3 4} Such evidence, while consistent with a causal effect of giving up smoking, may also be explained in other ways. The sort of person who might stop smoking may differ from the sort of person who is likely to continue—for

example, constitutionally^{5 6}—so that the differences observed in mortality may reflect differences other than smoking habits. That this explanation may have some substance was illustrated by a study showing that smokers who stopped smoking had, while they were still smoking, characteristics suggesting a substantially smaller risk of dying from coronary heart disease than comparable smokers who continued to smoke.⁷

Another way of trying to assess the effect on health of stopping smoking is to study whole populations who have greatly reduced their smoking and compare their trends in mortality with those of "otherwise similar" populations who have not. While not providing conclusive evidence (for which a randomised trial would be needed in which one group is forbidden to smoke while another continues to smoke), this method has fewer theoretical objections than the other type of study. Nevertheless, in such studies death rates from all diseases should be considered and not just those known to be smoking-related. If a person stops smoking he may do something else, and if this is hazardous to health the risk of other diseases, not necessarily conventionally related to smoking, may rise. This may be particularly important in high-need smokers,⁸ who may smoke to relieve stress. Smokers who smoke for physiological reasons are presumably less likely to stop than other smokers. Nevertheless, if such smokers are forced to give up by professional persuasion, legislation, or social pressure it is important to know whether their health is likely to benefit on balance.

British male doctors have reduced their smoking dramatically. This became clear from the study of Doll and Hill,⁹ in which 34 440 men, representing 69% of all British doctors, completed smoking questionnaires at intervals from 1951 to 1971. In 1951 these doctors smoked on average 9.1 cigarettes a day—that is, 88% of the amount smoked by UK men of the same age. By 1971 the figure had fallen to 3.6, which was only 37% of the national average.³ Their study also noted the cause and time of death of any doctor dying during the 20 years, and Doll and Peto³ tested for several groups of causes of death whether the mortality of the doctors studied had improved relative to the population at large.

These workers found statistically significant reductions in death from lung cancer at all ages, ischaemic heart disease and myocardial degeneration at ages below 75, and "other vascular causes associated with smoking" (including cerebral thrombosis) below the age of 65. Although they noted that with regard to coronary heart disease they were "probably chiefly observing the secular tendency for decline in the standardised mortality ratio in social class I, some of which may have been due to

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